Introduction

Catheter ablation in patients with recurrent VT requires identification of components of the reentrant circuit and is mostly limited to hemodynamically tolerated monomorphic VTs. However, most of the induced VTs are unstable, with multiple morphologies, and do not permit extensive pacing maneuvers during arrhythmias.

High-density electroanatomical mapping provides an accurate 3-dimensional (3D) characterization of the diseased myocardium and allows a substrate-based ablation strategy focuses on the identification of abnormal myocardium that participates as critical components of the reentry circuit. Pathological studies have suggested that zones of slow conduction/isthmus of the reentrant circuits are often located within myocardial scar, and EGM recordings from these sites often exhibit multi-component, delayed signals of higher voltage amplitude compared to the surrounding scar (Figure 43.1).\(^1,2\) By careful analysis of the electroanatomical substrate and local voltage profiles, VT-related conducting channels can be identified that correspond to the zone of slow conduction / isthmus site within the reentry circuit.\(^3,4\) Such channels are defined as paths demonstrating contiguous electrograms with voltage higher than that of the surrounding areas, and participate in orthodromic activation during VT (Figure 43.2). Identification of such channels helps to localize the potential reentrant VT circuit and focuses additional mapping effort.

Preprocedural Planning

A thorough preoperative assessment can facilitate procedural planning for efficient mapping and improves ablation outcomes. Defining patients’ underlying arrhythmogenic substrate requires careful review of patients’ history and data. In patients with coronary artery disease and prior myocardial infarction, the reentrant VTs often originate from subendocardial sites of infarcted myocardium, adjacent to the dense scar.\(^5,6\) However, nonendocardial substrates in scar-related as well as in idiopathic VTs have recently been increasingly recognized.\(^7-9\) In patients with dilated nonischemic cardiomyopathy, confluent areas of abnormal low-voltage scars (epicardial ≥ endocardial) are often located over the basal lateral left ventricle near the valve annulus. A prior history of open-chest coronary bypass grafting or valvular surgery is important if a percutaneous epicardial approach is being considered,\(^10,11\) whereas a history of significant peripheral vascular disease, prior mechanical aortic prostheses, or mitral valvular prostheses may preclude a retrograde aortic or transseptal approach for endocardial intervention.

The presence of Q-waves on the ECG provides noninvasive clues for localization of prior myocardial infarction and the potential scar arrhythmia substrates in patients is ischemic cardiomyopathy. A useful ECG algorithm based on 12-lead ECG allows localization of “site-of-origin” for ischemic VTs with a reasonable (> 70%) predictive accuracy to guide further mapping efforts.\(^12\) In the absence of
Ablation of VT

Figure 43.1 Anatomical substrate post-myocardial infarction. The left panel shows a theoretical construct of intramural scar-mediated reentry with diastolic activity recorded in the isthmus (electrodes 1 through 5) before exiting the circuit (bold arrow) between 2 areas of collagen (blue) on trichrome staining of an experimental infarction.

The right panel shows an endocardial resection of infarcted papillary muscle near the origin of the VT. Dense fibrotic connective tissue (C) was surrounded by rims of residual myocardial fibers (R). This was represented by the middle schematic drawing with dotted areas indicated viable myocardial cells and black areas indicated fibrotic connective tissue. Extracellular recordings from site A to M during stimulation, demonstrating varying degrees of fractionation and conduction delay. Slow conduction perpendicular to the fiber direction in infarcted myocardial tissue is caused by a “zigzag” course of activation at high speed.

(Adapted from Tung R. Circulation 2011;123:2284, with permission; deBakker J. Circulation 1988;77:589, with permission; and deBakker J. Circulation 1993;88;915, with permission.)

Figure 43.2 Channel identification with voltage color gradient adjustment. A reentrant VT circuit (Panel A) is depicted by the illustrative cartoon of scar and multiple pathways. The circuit resides in the low-voltage scarred tissue. The baseline color gradient shows dense scar (red), normal tissue (purple) and the intermediate colored border zone (Panel B). The VT-related conducting channels represents the orthodromic activation during reentry (isthmus→exit→outer loop) and often corresponds to zone of slow conduction (Panel C). Such channel may be visible by adjusting the color threshold on a voltage map (Panel D). VT-related conducting channels is defined as a path demonstrating contiguous electrograms with voltage higher than that of the surrounding areas (Panel E).
monomorphic VT, PVC morphologies during sinus rhythm can also be useful to locate the generalized regions of VT origin or ascertain the possibility of Purkinje involvement. Furthermore, various QRS morphologic criteria have been used to distinguish endocardial from possible epicardial or non-endocardial sites of origins.13-15

However, the 12-lead ECGs of the spontaneous presenting VTs are often unavailable. Utilization of the stored electrograms (EGMS) from patients’ ICDs can be helpful in providing additional data. Comparison of the cycle lengths and the intracardiac “far-field” or “near-field” electrogram morphologies during induced VTs can be used to distinguish “clinical” VT from “nonclinical” arrhythmias. Although the spatial resolution of pace mapping based on ICD electrogram morphologies is inferior to that of 12-lead ECGs, they may be useful for determining whether an ablation catheter is located near the VT exit site. This may focus mapping efforts, streamline workflow, and improve ablation outcomes.16

Although ischemia does not cause recurrent monomorphic VTs, an adequate assessment of the ischemic burden is essential, particularly in elderly patients with history of ischemic heart disease and prior surgical/percutaneous interventions. From a procedural safety point of view, we have a low threshold to perform coronary angiograms to exclude potentially significant coronary arterial stenosis in this population with extensive structural heart disease.

Echocardiography is useful to identify regions of wall motion abnormalities, wall thinning, or aneurysm consistent with prior infarcts/scar, suggesting potential locations of arrhythmogenic substrate. Echocardiography also allows identification of intracardiac thrombus, which would preclude an endocardial approach. The presence of aortic or mitral valve stenosis may influence and guide retrograde aortic versus transseptal ablation approach. Patients with both mechanical aortic and mitral valves may require an epicardial or alternative approach.

Magnetic resonance imaging (MRI) with gadolinium-delayed enhancement has increasingly been utilized for localization of arrhythmogenic substrates in both patients with post-infarction VTs and those with nonischemic cardiomyopathy.17-19 Regional wall motion abnormalities and wall thinning (WT) detected on multi-slice CT were also correlated to low voltage/scar regions, almost exclusively located within 3 mm of the thinnest region20 and were inside the MRI-delayed enhancement areas.21 The integration of CT/MRI imaging and electroanatomic maps can be helpful to plan the appropriate mapping and ablation strategies.

Unique to VT mapping, ECG pattern recognition is crucial in localizing the VT exit and for pace mapping. Accurate placement of surface ECG electrodes is imperative. Erroneous ECG interpretations will lead to confusion, procedural delay, and failed outcome.

Our protocol recommends the use of the electroanatomical system for substrate mapping (Carto, Biosense Webster, Diamond Bar, CA). Care must be taken for placement of the reference patch to compensate for the dilated, leftward rotated left ventricles in most of our patients. The reference patch should be placed lower in the middle of the ventricular silhouette in the anterior-posterior projection to ensure proper registration of the electroanatomical navigational data. Although the St. Jude Ensite NavX system (St. Jude Medical, St. Paul, MN) may also be used to construct the voltage maps, identification of VT-related conducting channels using this system has not been studied.

For the real-time ICD EGM recordings, a device-specific junction box (for example, Medtronic, Minneapolis, MN) may be obtained from the vendor that connects the programmer to the EP recording system (GE Prucka, Marlborough, MA) in the analog channels for display.

In our laboratory, the majority of our patients with left ventricular VT ablation, the arterial access is through the right femoral arterial approach. In patients with significant peripheral vascular disease, a long sheath is used for better support. A separate, redundant femoral arterial access may be considered for hemodynamic monitoring and support (such as intraaortic balloon pump) in patients with severe ventricular dysfunction. The majority of our patients also undergo intracardiac echocardiographic (ICE) imaging during either endocardial or epicardial VT ablation procedures. The right femoral vein is accessed to accommodate an 8- or 11-Fr sheath for retrograde aortic approach. Patients with significant peripheral vascular disease, a long sheath is used for better support. A separate, redundant femoral arterial access may be considered for hemodynamic monitoring and support (such as intraaortic balloon pump) in patients with severe ventricular dysfunction. The majority of our patients also undergo intracardiac echocardiographic (ICE) imaging during either endocardial or epicardial VT ablation procedures. The right femoral vein is accessed to accommodate an 8- or 11-Fr sheath for the placement of a 10-Fr phased-array ICE catheter. Two right ventricular catheters are routinely placed at the RVA and near the HB. The RVA catheter marks the ventricular apex, and the His catheter marks the ventricular base, opposite the aortic valve.

For procedures performed using an epicardial approach, or in patients who may have VTs that originate near the mitral annulus, an additional CS catheter, inserted either from the right internal jugular vein or from the femoral vein, is used to outline the basal LV silhouette and to facilitate mapping.

Anesthesia

Due to the severity of the underlying structural heart disease, associated multiple comorbidities, and the long duration of the procedure, full anesthesia support is routinely used. The majority of our patients with scar-based VT who undergo ablation are under general anesthesia. Multiple episodes of poorly tolerated arrhythmias are often induced and require shock terminations. Among the many
advantages, general anesthesia helps to control patients’ discomfort, minimizes movement, and improves mapping accuracy. The disadvantage of general anesthesia is the abolition of the sympathetic tone for the compensatory vasoconstrictive response during rapid VTs. Nonetheless, close collaboration between the anesthesiologist and the electrophysiologist allows optimal hemodynamic management and respiratory support during the procedure.

Anticoagulation

Intravascular insertion and manipulation of catheters, creation of ablation lesions, activation of coagulation factors, and potential disruption of atherosclerotic plaques contribute to a risk of thromboembolism during and after catheter ablation. Patients with structural heart disease undergoing left heart catheterization have a risk of stroke or thromboembolism of ~1%. We recommend meticulous monitoring of the ACT every 20 minutes. The target ACT is maintained at ~300 seconds. Of course, systemic anticoagulation is prohibited if percutaneous epicardial access is anticipated.

Mapping

Most patients who undergo VT ablation have significant structural heart disease and multiple inducible ventricular tachyarrhythmias (mean 4 ± 3 VT morphologies). A hybrid approach is needed, combining both the conventional mapping techniques and the substrate mapping approaches (Table 43.1).22,23 A conventional mapping strategy consists of activation mapping and entrainment mapping, and both require sustained reentry and cannot be performed during poorly tolerated VT.

To optimize the result of substrate mapping during sinus/paced rhythm, areas of interest within the abnormal myocardium should be tagged, which helps to define the geometry of the circuit and its relationship to the underlying scar. Identification of conducting channels, along with other collaborative mapping strategies, helps to characterize the VT circuit (these include defining EUS, detecting LPs, and pace mapping [Figure 43.3]). Conventional mapping methods such as activation and/or entrainment/resetting response also complement the substrate mapping to confirm the functional significance of these conducting channels.

In any given patient, the mapping strategy has to be individualized. Detailed, high-density EGM recording is essential. The average number of sampled points per chamber should be a minimum of 150 to 200 for adequate definition of anatomy and EP characterization of the VT substrate. Our protocol recommends the use of the open irrigated radiofrequency ablation catheter (NaviStar ThermoCool, Biosense Webster, Diamond Bar, CA). By cooling the electrode-tissue interface, irrigated electrodes allow for delivery of greater power without significant rise of impedance or catheter tip temperature. This consistently produces deeper and larger lesions compared to standard solid electrodes, and is particularly important for relatively large circuits, and reentry pathways may be located deep in scarred myocardium.24,25 In addition, the smaller distal mapping electrode pair (3.5 mm with 2-mm spacing) allows better spatial resolution compared to the standard 4- or 8-mm tip. The use of contact force sensing catheter is also standard in our laboratory to assure adequate substrate characterization and ablation lesion formation.26

In order to identify the potential VT-related channels, we first must perform a detailed electroanatomical voltage map to characterize the local voltage profile of the VT substrate. Bipolar endocardial ventricular signals are recorded and filtered at 10 to 400 Hz on the Carto System, and the peak-to-peak signal amplitude of the bipolar EGM is measured automatically. A 3D anatomical shell of the cardiac chamber is constructed, and the EGM signals are coupled and displayed as color gradients on a voltage map. Such voltage maps may be registered onto a previously constructed anatomical shell from the ICE images using the CartoSound software. Valvular locations are tagged and excluded from the voltage analysis. Valvular sites are identified by fluoroscopic catheter tip positions that demonstrate simultaneous recordings of equal atrial and ventricular signal amplitudes. The voltage maps are then edited, and intracavitary points are eliminated.

The reference value for distinguishing normal and abnormal bipolar EGM amplitude has been previously established at 1.5 mV for the right ventricle and 1.8 mV for the left ventricle. The normal signal amplitude is defined as the value above which 95% of all bipolar signal voltages from the endocardium of normal ventricles are included. “Dense scar” is arbitrarily defined as areas with signal amplitude less than 0.5 mV. The “border zone” is defined as a transition zone between dense scar and normal tissue (0.5 to 1.5/1.8 mV).27 For epicardial mapping, the “normal” epicardial signal amplitude is set at above 1.0 mV.

Isthmus sites have been shown to reside predominantly (> 80%) in the dense scar (< 0.5 mV) whereas most exit

### Table 43.1 VT mapping techniques

<table>
<thead>
<tr>
<th>Conventional Mapping Techniques</th>
<th>Substrate Mapping Techniques</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sinus rhythm mapping</td>
<td>Local electrogram voltage/amplitude</td>
</tr>
<tr>
<td>ECG analysis, pace mapping</td>
<td>Conducting channels (CC)</td>
</tr>
<tr>
<td>Activation mapping</td>
<td>Electrical unexcitable scar (EUS)</td>
</tr>
<tr>
<td>Entrainment mapping</td>
<td>Electrograms with isolated delayed components (E-IDC) or late potentials (LPs)</td>
</tr>
<tr>
<td>Personnel</td>
<td>Pace mapping</td>
</tr>
</tbody>
</table>

...
sites are located in the border zone (Table 43.2).4 Pace mapping along the border zone (0.5–1.5 mV)28 is performed to approximate the exit of the VT circuit, which is defined by sites with a similar paced QRS morphology compared to that during spontaneous ventricular arrhythmia with a short stimulus-QRS interval, arbitrarily defined as < 40 ms. Once the exit is identified, pace mapping and further mapping efforts should be directed progressively away from the abnormal border zone toward the center of the low-voltage dense scar (< 0.5 mV) for localizing the isthmus of the reentry circuit.29

Identification of Conducting Channels (CCs)

The Carto System displays the voltage maps and automatically selects the largest local component of the bipolar EGMs. The nominal setting for the color display of voltage maps has an upper threshold of 1.5 to 1.8 mV (purple) and a lower threshold of 0.5 mV (red). The border zone is the transition area between dense scar and normal tissue with intermediate colors.

Focusing on the low-voltage areas (<1.5 mV), VT-related conducting channels can be identified by voltage color adjustment of the bipolar voltage maps during baseline rhythms without tachycardia induction. A “conducting channel” is defined by the presence of a “corridor” of consecutive EGMs differentiated by the higher signal voltage amplitudes than the surrounding area.3 A conducting channel is also characterized by (1) multi-component, often delayed, signals such as local abnormal ventricular activities (LAVA) recorded within the channels and (2) local capture with a long stimulus-QRS interval.

With the current versions of the software (Carto 3), color threshold enhancement can be performed by manually adjusting the color bar. First, the upper threshold of the color display is reduced toward 0.5 mV to minimize the intermediate colors and to maximize the color contrast between adjacent myocardium with different EGM voltages. Thereafter, both the upper and the low-color thresholds are decremented in small steps (0.1–0.5 mV) until a conducting channel could be identified or a minimal value of 0.05 mV was reached for the lower limit of the color range. A “scar” may also be designated and displayed as a solid gray area based on the specified voltage value by using the “scar setting” software feature (Figure 43.4).

**Table 43.2** Local electrogram amplitude for sites within the reentrant circuit

<table>
<thead>
<tr>
<th></th>
<th>Entrance</th>
<th>Central Isthmus</th>
<th>Exit</th>
<th>Outer Loop</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dense Scar (&lt; 0.5 mV)</td>
<td>17</td>
<td>30</td>
<td>18</td>
<td>6</td>
</tr>
<tr>
<td>Border Zone (0.5–1.5 mV)</td>
<td>2</td>
<td>7</td>
<td>26</td>
<td>18</td>
</tr>
<tr>
<td>Normal (&gt; 1.5 mV)</td>
<td>---</td>
<td>---</td>
<td>4</td>
<td>8</td>
</tr>
<tr>
<td>Total (136 sites)</td>
<td>19</td>
<td>37</td>
<td>48</td>
<td>32</td>
</tr>
</tbody>
</table>

(Modified with permission from Hsia et al., Heart Rhythm. 2006;3, 503–512.)
It is important to note that the scar tissue is not homogeneous and there is a wide range of scar definition/voltage thresholds for best identifying the conducting channels. A single cutoff voltage threshold may not be feasible in all cases as VT-related complete (VTR-CCC) and incomplete (VTR-ICC) conducting channels are visualized at multiple voltage thresholds (Figure 43.5).  

The majority of such VT-related channels are located in scar areas with bipolar voltages between 0.2 and 0.3 mV, and can be identified noninvasively by contrast-enhanced magnetic resonance imaging. Such channels can be identified in 75%–88% of patients and have been correlated to the critical isthmus sites of VT circuit by entrainment mapping. However, the mere presence of voltage channels has a low specificity in predicting the location of the VT isthmus and additional mapping effort is needed to establish its functional significance. Evidence of delayed impulse propagation into the channels should be examined, which suggests the presence of a protected corridor of slow conduction.
Chapter 43: How to Use EAM to Identify Critical Channels for VT Ablation

Figure 43.6 Identification of VT-related conducting channel. Endocardial voltage maps with VT-related conducting channels in a patient with a large anterior myocardial infarction and incessant left bundle branch block-right-inferior (LBB-RI) monomorphic VTs.

Panel A: Endocardial voltage map shows a large anterior scar. The color gradient adjustments correspond to 0.5–1.6 mV, 0.35–0.55 mV, and 0.21–0.31 mV. A VT-related conducting channel can be identified at a color threshold at 0.21–0.31 mV.

Panel B: Decremental conduction delay within the channel was observed. Progressively prolonged stimuli to late potentials (LP)/local abnormal ventricular activities (LAVA) intervals (arrows) were demonstrated with faster pacing rates or delivery of ventricular extrastimulus. In addition, there was delayed activation of the second component of the fractionated local electrograms with delivery of premature ventricular extrastimulus. Panel C: Pace mapping facilitate the detection of conducting channels. Voltage maps show progressively delayed LP recordings with decremental slow conduction from the border zone toward into the scar during RVA pacing. Pace mapping within the channel (arrow) demonstrated a perfect pace map compared to that of spontaneous LBB-RI VT.
Ablation of VT

Figure 43.7  Identification of epicardial VT-related conducting channels. Epicardial voltage maps with VT-related conducting channels in a patient with an arrhythmogenic right ventricular cardiomyopathy (ARVC), recurrent monomorphic VTs, and a prior failed endocardial VT ablation. **Panel A**: Epicardial voltage maps over the right ventricle in the left anterior oblique (LAO) view. A channel is visible after color threshold adjustment from 0.5–1.5 mV to 0.5–0.8 mV. **Panel B**: Late potentials (LPs) are recorded within the putative VT-related channel during sinus rhythm, which becomes more obvious during RVA pacing at 600 ms. In addition, spontaneous oscillation of the QRS-LP intervals (arrows) are noted at the distal recording electrode pair, but remains stable at the proximal electrodes. This suggests decremental conduction delay between the proximal and the distal recording electrodes during propagation of activation wavefront from the RV apex. **Panel C**: Demonstration of decremental conduction delay within the channel. Progressively prolonged QRS-LP intervals and 2-to-1 conduction block with faster pacing rate. The voltage map shows progressively delayed LP recordings deeper into the channel during RVA pacing. Pace mapping within the channel demonstrated a perfect pace map with a long stimulus-to-QRS interval compared to that of spontaneous VT.
During sinus rhythms, delayed local activation (late potentials), or local abnormal ventricular activities (LAVA) recorded within the voltage channel may reflect slow impulse propagation into the isthmus. Such abnormal signals within the channels may be better identified during ventricular pacing compared to sinus rhythm, with a change in the direction of the activation wavefront that unmasks areas of functional block or conduction delay. Introductions of ventricular extrastimuli can also reveal local decremental conduction delay into a channel (Figure 43.6 and Figure 43.7). The presence of late fractionated potentials/LAVA within the voltage channel significantly increases the specificity for identifying the clinical VT isthmus, especially at sites with significantly prolonged local conduction delay (QRS-LP interval > 200 ms). Commonly, only a single channel is observed that corresponds to the potential reentrant isthmus site by pace mapping. By functional block, the conducting channels can also be correlated to the VT isthmus site by pace mapping. By careful analysis of the QRS matches during pace mapping, entrance and exit of the isthmus can be identified. Selected pace mapping with local capture within the conducting channel should produce a QRS similar to that of VT, often with a longer stimulus-to-QRS interval (S-QRS) due to slow conduction within the channel (Figure 43.8).

Electrical unexcitable scar (EUS) is characterized by non-capture with high-output pacing (>10 mA) and is associated with a very low local bipolar voltage (<0.25 mV). EUS identifies non-conducting tissue that often borders viable myocardium during sinus rhythm and is commonly located in close proximity to the VT isthmus and conducting channels.

With emergence of multi-electrode mapping catheters/balloon, the standard criteria for identifying low-voltage scar and channels may not apply to these newer-generation mapping tools. The local electrogram voltages are dependent on the recording electrode size, inter-electrode spacing, and the angle of wavefront propagation. Comparing the standard ablation catheter with the 3.5-4.0 mm distal electrode, myocardial scar characterization using the small (1.0 mm), closely spaced electrodes (PentaRay®, Biosense Webster) resulted in a 22% smaller scar area (<1.5 mV), with a 47% reduction in dense scar size (<0.5 mV) in an animal model of infarction. High-density recordings may help to identify heterogeneity within the area of low voltage, allowing better localizing channels of surviving myocardial bundles, with significantly more fractionated electrograms or LAVA detected within these channels (Figure 43.9). However, prospective studies comparing high-resolution, multi-electrode mapping with point-by-point, standard-size electrode recording are needed to establish new criteria/thresholds to define VT-related conducting channels, to characterize the potential isthmus sites, and may provide further insight into the electroanatomical substrate of scar-based reentrant VTs.

Ablation

The primary goal of catheter ablation of scar-related VT is the interruption of critical isthmus region. Most patients with scar-related VT present with unstable arrhythmias that are not amenable to point-by-point conventional mapping techniques based on intracardiac activation sequence and the response to entrainment mapping. Ablation strategies targeting the abnormal electroanatomical substrate have reported improved arrhythmia-free survivals at short- and mid-term follow-up.

After visually identifying the potential VT-related channels, the functional significance of the channels should be confirmed by activation mapping and/or entrainment/resetting response (Figure 43.10). Locations of interest within the abnormal myocardium are tagged to define the geometry of the circuit and its relationship to the underlying scar. This is important in facilitating the design of the ablation lesion set(s). Linear ablation lesion sets are performed to transect the channel/isthmus or to connect the VT exit sites to anatomical barriers such as the EUS or the valvular annuli.

The conducting channels identified within the scar are often interconnected. Ablations targeting the entrance of channels with relatively early abnormal potentials often result in delay, partial, or complete eliminations of neighboring and remote late potentials/LAVAs within the substrate. Scar de-channeling focuses ablations targeting the entrance sites of such channels, aiming towards the end point of eliminating a consecutive series of late potentials/LAVA (Figure 43.11). Improved event-free survival rates were observed in patients that were non-inducible after scar de-channeling and those with complete elimination of EGMs within the conducting channel. Arrhythmia recurrences are mainly related to incomplete conducting channel-electrogram elimination. Given the limitations of programmed stimulation, the overall strategy of scar de-channeling, and targeting abnormal potentials significantly reduces VT recurrence rates. Freedom from ventricular arrhythmia after catheter ablation is also strongly associated with an improved transplant-free survival.

Epicardial Approach

In general, epicardial mapping is similar to that of the endocardial surface, with some noticeable caveats. First, the normal epicardial bipolar voltage is identified as >1.0 mV. Second, care must be taken to distinguish true scar from epicardial fat or poor contact/coronary arteries. Measurements of abnormal EGMs should demonstrate not only low amplitude but also discrete multicomponent, broad/split signals, and/or LPs (Figure 43.7). Third, pace...
Ablation of VT

Figure 43.8  Identification of the conducting channel along the mitral annulus. Panel A shows the bipolar endocardial voltage maps in a patient with an inferior myocardial infarction and recurrent VT. Two morphologically distinct VTs were induced: a RBBB-right-inferior (RBRI) QRS VT and a LBBB-left-uperior (LBLS) QRS VT. On the left, the voltage map is depicted by the standard color range, 0.5–1.5 mV, are shown. On the right, after the voltage threshold adjustment to 0.49–0.5 mV, the purple highlighted in the area greater than 0.5 mV and the red depicted areas with voltage <0.49 mV. The conduction channel with high-voltage area was clearly identified along the inferior scar mitral annulus. A potential reentry isthmus was defined between the dense inferior scar and the valvular annulus. Panel B shows confirmation of bi-directional conduction block with pace mapping before and after linear ablation to transect the mitral annular isthmus.

Before ablation, pace map at the site near the septum (site 4), revealed a good QRS match to the LBLS VT with short stim-QRS interval, 46 ms, as the exit site. Pace map near the basal lateral wall (site 1) shows a similar paced QRS morphologies to that of the RBRI VT with a short stim-QRS interval of 44 ms. However, pace map deep in the isthmus (site 3 and 4) revealed variable degrees of fusions with poor matching QRS morphologies, suggesting wavefront propagations out of both exits sites.

After ablation, pace map just septal to the ablation line (site 3), deep in the protected isthmus, produced a perfect QRS match to the septal exiting LBLS VT with a long stim-QRS interval (96 ms). However, pace map at the site just lateral to the ablation line (site 2, a few millimeter from site 3), shows an abrupt change in the paced QRS morphology with long stim-QRS interval (168 ms). This paced QRS morphology was identical to the RBRI VT exiting from the lateral side of the scar. Compared to pace mapping results before the ablation, these findings suggested the presence of bidirectional block at the isthmus along the mitral annular circuit.

RBBB: right bundle branch block morphology (positive) in V1; LBBB: left bundle branch block morphology (negative) in V1.
mapping or entrainment mapping with local capture may be difficult because of the elevated pacing threshold in the epicardium. Epicardial scar de-channeling has also been shown to be effective in patients with arrhythmogenic right ventricular dysplasia cardiomyopathy (ARVD/C).48

**Future Development**

Improvements in high-density mapping and software further help to identify conduction channels in VT substrate. Ripple mapping is a novel method that integrates local bipolar electrogram voltage with simultaneous wavefront propagation on a 3D anatomical geometry (Carto3v4, Biosense Webster). Using a multi-electrode catheter (PentaRay®, 2-6-2 mm spacing), coupled with a continuous automated point collection module (ConfIDENSETM, Biosense Webster), ripple mapping displays each electrogram at its 3D coordinate as a bar changing in length according to its voltage–time relationship.

Ripple mapping can identify slow conduction channels as delayed “ripples” propagating into the ventricular scar to guide ablation. During hemodynamically stable VT, ripple mapping may identify diastolic pathways contained within the confines of the conduction channels.49 Ablation of conducting channels using ripple-mapping algorithms has been shown to be promising. Ventricular tachycardia was non-inducible in 85% of patients postablation, and 71% remain free of VT recurrence at 6-month median follow-up.50 Persistence of such ripple mapping conducting channels was sensitive for VT recurrence but the specificity may be limited by blind-alley channels.
Ablation of VT

Postprocedure Care

Standard postoperative care protocol is recommended. Protamine may be given to help reverse the heparin, and access sheaths can be pulled once the activated clotting time is less than 180 seconds. In our laboratory, femoral arterial access is usually closed using the Perclose ProGlide suture-mediated closure system (Abbott Vascular, Pawtucket, RI). A large net-positive fluid balance is expected when using irrigated-tip catheters for endocardial VT ablations. Additional diuretics are often required in patients with advanced ventricular dysfunction.

In patients who underwent left ventricular endocardial ablation procedures, postoperative anticoagulation is recommended for the duration of ~1 month. Oral warfarin or target specific oral anticoagulants (TSOACs) can be started the day of the procedure for stroke prevention and minimizing thromboembolic complications. Lovenox, or low-molecular-weight heparin, is generally not required to bridge these patients. In all other patients (RV ablation, epicardial ablation, patients who are poor candidates for anticoagulation) aspirin 325 mg daily is recommended.

For epicardial VT ablation procedures, we generally leave a 5-Fr pigtail catheter for overnight drainage in the epicardial space. Follow-up transthoracic echocardiograms are performed after the procedure and at next day. We routinely administer steroid (Triamcinolone 2 mg/kg, diluted in 10–20 cc) into the epicardial space, with the pigtail catheter clamped for the first 4 hours before opening to suction drainage.

Procedural Complications

Catheter ablation of VT is a complex intervention usually performed in patients with advanced heart disease. The incidence of major procedure-related complications reaches 8%, with up to 3% procedure-related mortality, often due to incessant VT.

In order to reduce procedural complications, we try to minimize arrhythmia induction and procedural time. A hybrid approach is used with emphasis on substrate characterization during sinus/paced rhythm. Detailed and meticulous mapping techniques are essential to identify surrogate markers of slow conduction (channels, LPs, pace mapping) with limited VT induction.

Limitations

Since the CARTO system automatically selects the largest local component of the bipolar electrograms, care must be given to measure the appropriate “local” signals and to exclude far-field recordings or pacing artifacts. Sufficient sampling is essential for detecting voltage channels and additional mapping (pace mapping, activation/entrainment...
mapping, LAVA assessment) is necessary to establish its functional significance in VT circuits.

Using the standard 3.5-mm tip electrode, VT-related conducting channels in scar substrate can only be identified in ~70% of patients with monomorphic hemodynamically stable VTs. This may be due to (1) portions of the reentrant circuit were intramyocardial or epicardial, (2) the reentrant circuits were largely determined by functional rather than fixed anatomical barrier, or (3) signal averaging effect from far-field adjacent healthy myocardium. A slow VT with circuit resides in dense scar with significant conduction delay may favor the identification of critical channels, whereas nonuniform, nonsustained rapid VTs may involve a smaller isthmus and narrower entrance-exit points with minimal low-voltage area without dense scar. Identification of VT-related channels thus may not be feasible in those patients.

Although high-density multi-electrode minimizes the effects of far-field tissues, facilitates LAVA detection, and improves mapping resolution, more experience is needed to establish new criteria for identification of voltage channels and characterizing the VT substrate, especially in patients with nonischemic cardiomyopathies with mid-myocardial substrate.

Figure 43.11  Scar de-channeling. Panel A: Inferior view of bipolar voltage substrate maps during sinus rhythm before (MAP) and after (reMAP) scar de-channeling in a patient with prior myocardial infarction. Electrograms recorded as conducting channel entrances are labeled with black dots and inner sites with blue dots. Examples of bipolar electrograms at entrances (1 and 5) and inner parts (2–4) are shown (left). Delayed components of the electrograms are highlighted with arrows. Electrogram aspect after elimination of the delayed component (asterisks) in the same sites after scar dechanneling is shown (left). MA indicates mitral annulus; and LV, left ventricular. Panel B: Kaplan–Meier curve for the primary end point of any sustained ventricular arrhythmia or sudden cardiac death according to the complete/incomplete elimination of conducting channel and the need for residual VT ablation. (Modified from Berruezo A, Fernandez-Armenta J, Andreu D, et al. Circ Arrhythm Electrophysiol. 2015, 8(2):326-336, with permission.)

Summary

The anatomic extent and location of components of reentrant VT circuits can be defined by detailed electro-anatomical mapping. The anatomic size of the isthmus of slow conduction for VT is at least several centimeters long, with isthmus sites typically identified within densely scarred myocardium (0.2–0.3 mV). Conducting channels that correspond to zones of slow conduction within the circuit can be identified by adjusting the color thresholds of the display bipolar voltage maps made during sinus or paced rhythms in most patients. Identification of conducting channels should be considered as a major strategy for substrate mapping and ablation of VT, particularly in those patients with large, dense myocardial scar (either endocardial or epicardial).

To optimize the substrate mapping result during sinus/paced rhythm, multiple techniques are utilized to define areas of slow conduction as surrogate markers for the potential reentry circuit isthmus. Limited VT induction with activation or entrainment mapping may be performed for EP confirmation of the isthmus location. Placement of linear ablation lines, designed to transect preferred channels of conduction in more densely scarred regions of abnormal myocardium, may facilitate ablation
of multiple stable and unstable VTs, even in the absence of VT induction.

References

Chapter 43: How to Use EAM to Identify Critical Channels for VT Ablation


